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Microscopical Studies in Pelvic Peritonitis

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MICROSCOPICAL STUDIES IN PELVIC PERITONITIS.[1]

PROBABLY no disease is more frequently met with by gynecologists than pelvic peritonitis, and few have been more carefully studied as to causes, symptoms, treatment, sequelæ, etc.

From a clinical stand-point, I have long been convinced, as I said in an article published in the Pittsburg *Medical Review*, October, 1889, that in plastic peritonitis, when the tubes and ovaries are bound in with pseudo-membranes, as also in purulent peritonitis, when there are larger or smaller abscesses, in connection with, and as a result of, disease of the tubes and ovaries, the only way of permanent cure is by removal of the diseased structures.[2]

To find if possible a better way, and to learn more of the nature of the disease or pathology of the conditions, I have for months past been making, with the microscope, researches in the morbid changes, the minute anatomical features of pelvic peritonitis, and now present the results of my investigations. I am not aware that anyone has before studied the subject microscopically.

To secure definite results in the microscopical examination of any tissue, the first necessity is a proper preparation. After working for more than fifteen years in microscopy, I am convinced that the best method is: First, harden the specimens in one-half of one per cent. of

[1] Pursued in Dr. Charles Heitzman's laboratory.

[2] William Duncan says, in the British Medical Journal: "Nothing short of abdominal section will cure. The old method of treating dilated tubes by tapping per vaginam, or by electricity, is not only unscientific, but more dangerous than abdominal section." Doran, in the same journal, says: "Abdominal section is more satisfactory, and even safer, than palliative measures."

chromic acid, which gives a distinctness that cannot be secured by any other preparative fluid; then, after thin sections are made, mount in chemically pure glycerine, and not, as is the usual way, in Canada balsam, which so clears up the specimen that it cannot be satisfactorily studied with high powers of the microscope; while specimens mounted in pure glycerine, after previous hardening in chromic acid, are fit for the finest definition by the highest powers of the microscope.

The almost universal method of mounting in Canada balsam has probably put back the progress of pathological investigations more than twenty years. Recently some microscopical slides were sent me from a well-known university. They were beautifully mounted, but done in Canada balsam, and I could scarcely recognize my old friends, endothelioma and gyroma; and certainly from specimens so prepared I could never have defined and learned the peculiar changes characteristic of these two diseases.

In this article I will give, first, a description of the normal peritoneum as I have studied it microscopically, and note certain anatomical facts which seem to have escaped the attention of previous observers; second, the pathology of inflammation; third, describe the morbid changes of peritonitis in the various constituent parts of the peritoneum, viz., endothelium, connective tissue, blood-vessels, and smooth muscles; and fourth, the etiology of peritonitis.

The peritoneum is a connective-tissue formation, closely attached to the organs of the abdominal cavity, most of which are entirely, others partially, covered by it. The free surface of the peritoneum is covered with a layer of so-called endothelium (Fig. 1).

Endothelia are polyhedral, nucleated, protoplasmic bodies of greatly varying sizes, and separated from each other by a narrow, light ledge of cement substance. They are claimed to be connective-tissue formations by German and English histologists. The distinction between endo-

thelia and epithelia is artificial and lacks logic and consequence. They are similar in structure and functions, and appear alike under the microscope. At the fimbriated extremity of the tubes they unite and are continuous; also in the liver the epithelia join the endothelia of the capillaries, and are in direct connection with them. Waldeyer has shown the covering layer of the ovary to be

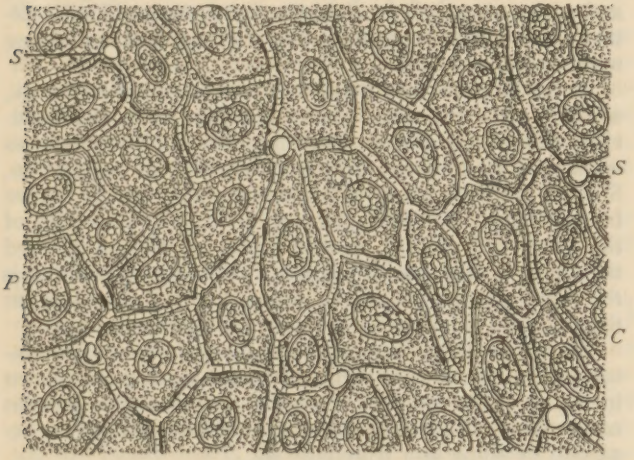

FIG. 1.—Normal Endothelium of the Peritoneum Covering the Tube. (× 1,000. Front view.) *P*, polyhedral nucleated protoplasmic bodies; *C*, cement substance, traversed by delicate spokes; *S S*, stomata.

true epithelium, which he traces to the embryonal development of the germinal epithelia.

Nitrate of silver renders the cement substance of both endo- and epithelia dark brown. These dark brown lines, if viewed under high powers, appear dotted and interrupted, the light interruptions corresponding to delicate thorny projections of the protoplasmic bodies, traversing the cement substance, and known as thorns or "prickles" for the last thirty years. They are a contin-

uation of the reticulum of living matter within the epi- and endothelia, and represent the inter-communicating links between the single epi- or endothelia.

The fine net-work of living matter in each endo- and epithelium gives to the little formations their wonderful vital properties. These apparently shapeless masses of protoplasm perform some of the most vital physiological functions. All gland structures are formed of epithelia, and every secretion is elaborated by them. Each epithelium is a laboratory in itself, selecting and combining with marvellous skill.

In the kidneys they make a most complex structure, working with the same exact precision. In the bronchial tubes the millions of moving cilia do their ceaseless work, and so help in the great function of respiration. Similar epithelia carry safely the embryo of the future being through the Fallopian tubes. In the ventricles of the brain, and in the central canal of the spinal cord they have a like useful purpose. Equally marvellous is the work of the endothelia lining the blood-vessels, or the millions which cover the vast peritoneal membrane.

We notice in the cement substance, at some of the intersections of the endothelia, openings or rings, as shown in Fig. 1. The stomata or stigmata are the open mouths of the lymphatics, since the whole peritoneal cavity is regarded by some as a lymphatic sac.

Beneath the endothelia of normal peritoneum is the layer of fibrous connective tissue, which is divided into two portions. The one nearest to the endothelia is made up of more delicate fibres, and abounds in capillary blood-vessels, whereas the deep layers are built up of coarse bundles, and hold arteries, veins, and capillaries, but a scantier supply than the upper layers. In the peritoneum of the broad ligament are found many smooth muscles from the ligamentum teres, which is largely composed of smooth muscle-fibres, and blends with the connective tissue of the various organs of the peritoneum.

Inflammation.—Pathologists have held varied and op-

posing views as to what *inflammation* is, and what are the minute anatomical changes in the morbid process. For twenty-five years the theory of Cohnheim, that inflammation is an emigration of colorless blood-corpuscles, has been generally accepted, and is still maintained by many of the best pathologists, though it has been repeatedly disapproved. Cohnheim held that inflammatory corpuscles were an aggregation of emigrated white corpuscles, and that the tissue took no part in the inflammatory process.

It is true that the most intense inflammation is near the blood vessels, the source of nutrition, and consequently there we always find the greater number of inflammatory corpuscles or protoplasmic masses. But does it therefore follow that these masses of living matter, many of which are considerably larger than the calibre of the vessels, have emigrated from the vessels?

We frequently find nests of inflammatory corpuscles where muscles or other tissues have broken down, and yet there are no blood-vessels in the immediate proximity. Even if these masses were emigrated leucocytes, could these emigrated leucocytes break down the tissues? As I have studied inflammation with high powers of the microscope I could never recognize inflammatory corpuscles as extravasated leucocytes. The most conclusive proof of the incorrectness of Cohnheim's theory is seeing the blood-vessels themselves breaking down into inflammatory corpuscles.

Inflammation, by the humeral pathologists also, was regarded as a disturbance of the vascular system. Rokitansky held that inflammation was a disease of the blood, that inflammatory and pus corpuscles originated from the exuded plasma, and this exudate became organized and produced pseudo-membranes, or hyperplasia of inflamed tissues. Virchow recognized that some tissues, such as cornea and cartilage, though devoid of blood-vessels, were often the seat of inflammation, hence he asserted that the blood-vessels took but little part in the inflammatory process, but that the latter essentially con-

sisted in division and subdivision or proliferation of the stable or fixed cells of the connective tissue, from which originated pseudo-membranes, hyperplasia, and pus. But even Virchow's theory does not explain or account for the presence of the multitude of new elements in an inflamed tissue, which could not result simply from a division and subdivision of the cells.

Attention has recently been called to the complex changes of the nuclear stroma, leading to a division of the nucleus, which changes are called indirect division by Karyokinesis, or Karyomitosis (Klein). This theory confirms at least that the cells multiply and assume varied forms, and it has induced many German pathologists practically to abandon the theory of emigration, and to return to the original views of Virchow.

If we study the inflammatory process under the microscope, we can see clearly that the tissue itself breaks down into protoplasm, that there is an increase of living matter, and some of the coarse granules form homogeneous lumps, or large masses of protoplasm, from which are developed inflammatory corpuscles.

I have watched these changes in various tissues, repeatedly seen them in the peritoneal membrane, traced the increase of living matter, the multiplication of tissue elements, and the formation of the inflammatory corpuscles. This increase of living matter is a natural consequence of the larger supply of blood, corresponding to the familiar and clinical description of "redness and swelling."

These newly formed protoplasmic masses or inflammatory corpuscles, when examined under one thousand or twelve hundred power, show a fine reticulum, and we also see they are interconnected by thorny projections or delicate conical offshoots through the cement substance.

As early as 1873, Dr. Charles Heitzman showed that the basis substances of connective tissue are endowed with life, the same as the protoplasmic bodies themselves. When from some irritation there is a liquefaction of the

solid parts of the basis substance, and the protoplasm therein is set free, there will also be in this protoplasm an increase of living matter, just the same as in the epi- or endothelia, or in the original protoplasm of the so-called "cells." The protoplasm sprung from the "cells" and that from the basis substance is the same, and both alike develop into inflammatory corpuscles. If the latter remain interconnected, they gradually become spindle-shaped, and by a reformation of basis substance new connective tissue is produced. This constitutes "plastic" or "formative" inflammation. If, on the contrary, the connections or offshoots between the protoplasmic masses is broken, then inflammatory corpuscles become pus corpuscles, and suppuration commences, resulting in destruction of tissue. Pus is not tissue, and cannot produce tissue.[1]

Professor Stricker, of Vienna, originally held that all inflammatory corpuscles were pus corpuscles, and that inflammation and ulceration were identical. Every day clinical experience shows that only a limited number of inflammatory processes is suppurative; the greater majority are plastic and lead to an augmentation of the tissue involved. Since 1880 Professor Stricker has sustained the views as taught by Dr. Heitzman, that in so-called plastic or formative inflammation the newly formed inflammatory elements remain interconnected, thus being able to produce new tissue by a reformation of the basis substance; while if the connection is broken between the inflammatory corpuscles, they become pus corpuscles.

Recognizing these facts, which are borne out in the whole history of inflammation, the formation of pus corpuscles, and the difference between pus corpuscles and

[1] Hamilton, in his work on Pathology, says: "Suppuration is the separation from the organism by a natural process of death and degeneration of such inflammatory products as have accumulated within it." Senn, in his Principles of Surgery, says: "In suppurative inflammation of a serous membrane the leucocytes and embryonal cells are transformed into pus corpuscles, and in this way purulent peritonitis is produced."

inflammatory corpuscles becomes clear, simple, and comprehensive.

In studying peritonitis we see repeatedly the formation of protoplasm, the increase of living matter, the change to inflammatory corpuscles, the fine reticulum, the thorny projections between, or connecting links, and how, if the inflammatory corpuscles remain interconnected, they become spindle-shaped, and by a new formation of basis substance new tissue or pseudo-membranes are formed, which latter, if the inflammatory action continues, will increase and bind more firmly together neighboring organs in the peritoneal cavity. On the contrary, if the life connection between the living matter or inflammatory corpuscles is broken, they become pus corpuscles, abscesses are formed, or general suppuration commences, leading to more or less destruction of tissue.

Inflammatory Changes of the Endothelia.—"Cornil and Ranvier were the first who maintained that in inflammation the endothelia are enlarged, and their nuclei divide and become the source from which pus corpuscles are formed."

"The inflammatory changes of the endothelia of the peritoneum (omentum) has been studied more accurately by H. Kundrat (*Medic. Jahrbücher*, 1872). He notices, first, a disappearance of the cement substances, in place of which scattered globular bodies were seen. Next, enlargement of the nucleoli and the nuclei of the endothelia followed. In recent peritonitis he found large multinuclear bodies, which greatly surpassed in size single endothelia, and in purulent peritonitis a marked new formation of inflammatory corpuscles, but probably not all of these offshoots are the offspring of endothelia. Kundrat was the first to maintain that from the endothelia of the peritoneum, in chronic plastic peritonitis, connective tissue arises which leads to the formation of vegetations, and that this process takes place in two ways: either the endothelia themselves become spindle-shaped,

elongated, transformed into fibrillæ, or large nucleated protoplasmic layers, sprung from the endothelia, become directly fibrillated."[1]

In the mildest cases of peritonitis the inflammatory changes of the endothelia are the following (Fig. 2):

First, the cement substance, in consequence of some irritation, becomes liquefied. The endothelia are reduced to protoplasm; they become coarsely granular; there is an increase of living matter; the nuclei are enlarged and become more numerous, and the thorny formations of living matter of the cement substance are increased in size.

Many of the granules in the protoplasm of the endothelia unite and form homogeneous lumps; some of the enlarged endothelia fuse together and form multinuclear protoplasmic bodies, in which new lines of demarcation become visible, leading to the formation of inflammatory corpuscles. If all the granules and lumps of living matter and newly formed nuclei or inflammatory corpuscles remain interconnected by delicate offshoots, the result will be plastic peritonitis.

The endothelia are not, as Kundrat supposed, directly transformed into spindle-shaped bodies. They always split up into inflammatory corpuscles before a new formation of spindles takes place. As long as the layer of cement substance envelops the free surface of the endothelia, no agglutination can take place between contiguous peritoneal layers. The basis substance is first liquefied and sets free the living matter; then there is a multiplication and division of this living matter, inflammatory corpuscles are formed, they become spindle shaped, and then, by a formation of the basis substance, new tissues or pseudo-membranes are developed, which cause the agglutination of neighboring organs.

In suppurative peritonitis the living matter in the endothelia increases and divides and changes into inflammatory corpuscles, then the life connection between the

[1] Heitzman: Microscopical Morphology, p. 420.

inflammatory corpuscles is broken, they become pus corpuscles, and suppuration begins. Plastic peritonitis may

FIG. 2.—Acute Pelvic Peritonitis of Moderate Intensity. (× 500.) *E E*, endothelial layer; *F*, fibrous connective tissue; *C*, capillary blood-vessel.

be in one portion of the peritoneum, while suppurative peritonitis is progressing in another.

The Inflammatory Changes of the Endothelia Lining the Blood-vessels are exactly the same as in the endothelia covering the peritoneum. They become enlarged, coarsely granular, their nuclei augment and become increased in numbers. Sometimes the swelling of the endothelia of the blood-vessels, especially of the capillaries, causes a complete choking of the calibres. The capillaries are solidified and converted into solid tracts of fibrous connective-tissue. This enlargement of the endothelia by new formations of living matter, which afterward change to fibrous connective tissue, is the cause of endarteritis obliterans, leading to a narrowing of the calibre or a complete closure of the vessels. We often see in newly formed or inflamed tissue a new formation of blood-vessels, sometimes a complete vascularization, especially in pseudo-membranes of recent date.

The epithelia covering the ovaries act in the same way under irritation, and are subject to similar changes, first becoming coarsely granular, the living matter increasing, the nuclei dividing, a number of epithelia fusing together into multinuclear lumps, and then developing into inflammatory corpuscles. The latter, if the connection continues, form a new tissue, or pseudo-membranes, as mentioned above.

Inflammatory Changes of the Fibrous Connective Tissue.—In inflammation of this tissue the basis substance is first liquefied or melted out, and the bundles are transformed into more or less extensive fields of granular protoplasm, as seen in Fig. 2. The fibrous connective tissue immediately adjacent to the endothelia is almost entirely broken down into granular matter, while in the lower portion of the peritoneum there may be little change.

In more advanced stages of inflammation the original bundles of the fibrous connective tissue are reduced to spindle-shaped bodies, similar to those from which fibrous connective tissue has arisen in the early stages of its development (Fig. 3).

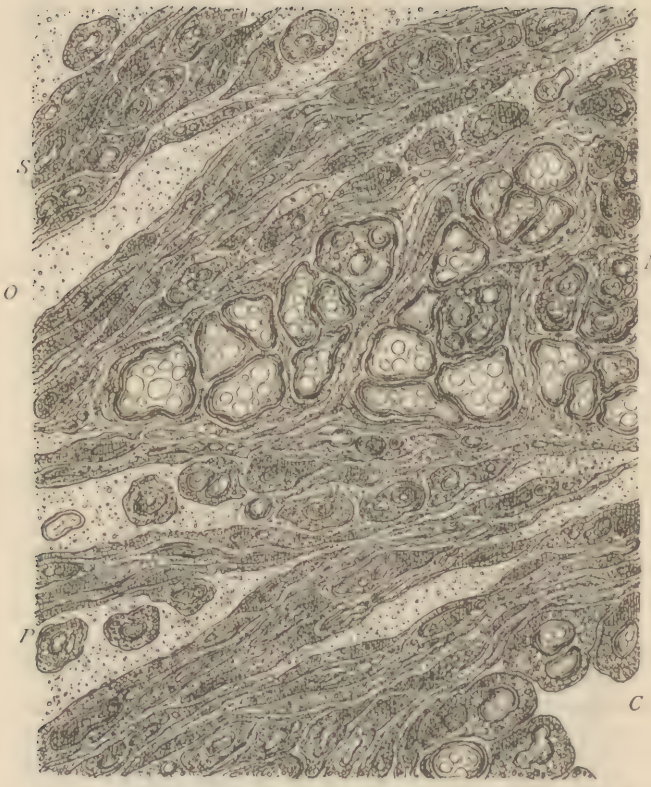

FIG. 3.—Intense Acute Peritonitis of the Broad Ligament. (×1,000.) *M*, transverse sections of smooth muscle-fibres, showing inflammatory changes; *S S*, bundles of fibrous connective tissue, broken up into spindle-shaped inflammatory corpuscles; *O*, œdema, serous exudate between the bundles; *P*, protoplasmic bodies, suspended in the serous exudate; *C*, capillary blood-vessel, showing inflammatory changes of the endothelia.

We see coarsely granular and nucleated spindles grouped together and retaining the original forms of the bundles. Between the groups are interstices of varying

diameter, filled with coagulated albumin, or albuminous fluid, forming œdema. The spindles constituting the original bundles remain interconnected. In the œdematous interstices are protoplasmic bodies, which probably have taken origin from emigration through the walls of the capillaries.

In the third and most intense form of inflammation the bundles are transformed into chains of homogeneous, highly glistening bodies, which result from a rapid increase of living matter, held within the bundles after the preceding liquefaction of the basis substance. (See Fig. 4.)

Such solid lumps are most numerous at the points of most intense inflammation. The more intense the morbid process, the greater the change of the tissues. Sometimes whole bundles are transformed into coarsely granular protoplasm, retaining their original size and shape, or breaking up into homogeneous lumps of living matter.

The free protoplasm between the bundles of fibrous connective tissue is the first to become changed into coarsely granular, freely nucleated masses, as in Fig 2. These clusters are seen especially in the deeper portions of the peritoneum, where there is comparatively a slight although progressive inflammatory process present. In the severest forms of peritonitis the difference between the inflammatory corpuscles sprung from free protoplasm and those from the previous bundles disappear, and the whole tissue is in a condition known to pathologists as inflammatory infiltration.

Examining these inflammatory corpuscles, derived from the fibrous connective tissue, with high powers, we see in them the same fine reticulum, and the corpuscles interconnected and connecting with the surrounding protoplasm by fine, delicate offshoots. This beautiful net-work of living matter, which we find in every part and in every organ, proves that the body is one complete whole, and not made up of independent "cells." It explains, too, that mysterious life-power, that inter-depen-

dence and sympathy by which one function follows another in such transcendent order.

When, from any cause, this connection between the in-

FIG. 4.—Intense Acute Tubal Peritonitis. (× 500.) *E E*, endothelial layer; *F*, fibrous connective tissue; *M*, smooth muscle-fibres in transverse section; *C*, capillary blood-vessel.

flammatory corpuscles is destroyed, as probably by certain microbes, the inflammatory corpuscles become pus corpuscles, and suppuration is established.

If, on the contrary, inflammatory corpuscles remain interconnected, they, after subsidence of irritation, become spindle-shaped, and, by reformation of the basis substance, form a new tissue; thus pseudo-membranes will be developed, and there will be a thickening or a hyperplasia of the peritoneum. This hyperplasia of the cellular tissue of the peritoneum, resulting from repeated attacks of inflammation, has been recognized as "cellulitis" or parametritis. Professor William Polk, in an article published in the MEDICAL RECORD, September 18, 1886, says: "Inflammatory masses under the headings of pelvic cellulitis and peritonitis are the results of salpingitis plus peritonitis."

A writer in the *Boston Medical and Surgical Journal* of July 9, 1891, gives an article on laparotomy, as he stated, verifying the diagnosis of "pelvic cellulitis." He says: "I found the tubes and ovaries in gross appearance healthy. I performed laparotomy, removing the tubes and ovaries of each side. Since the operation, six months ago, the patient has gained very much in general health and is free from pelvic disturbance of pain and discomfort." Evidently the patient had pyosalpinx, which caused the peritonitis and consequent adhesions.

In over eighty laparotomies I have never yet been able to find a mass of inflamed pelvic cellular tissue independent of the hyperplasia of the peritoneum, or the inflammatory adhesions around inflamed structures or diseased uterine appendages. With hand in pelvic cavity, I have searched repeatedly and carefully, yet have never been able to recognize an independent mass to be denominated "cellulitis."

Dr. T. G. Thomas, on December 21, 1882, when he reported his first four cases of the removal of uterine appendages, said, in referring to an article by Lawson Tait, published in the *British Medical Journal*, July 29, 1882,

that "it was one of the most valuable contributions to gynecological science made during the present decade," and added: "How often has it been found that, after a long course of treatment of the uterus, including all the mechanical devices and operative procedures that the most skilful gynecologist could devise, the condition of the patient remains substantially the same, and has finally to be abandoned as incurable."

Many physicians can verify that cases of so-called "cellulitis" have been treated for years without a cure being effected, while in other instances, if the cause of irritation or the source of infection be removed, the patient will in a short time be restored to health. As Dr. Grigg, President of the British Gynecological Society, in his opening address, said: "These methods have shed lustre upon gynecology as a curative art, and done much to raise it to a position approximating that of an exact science."

Inflammatory Changes in the Muscles.—Peritonitis presents an excellent opportunity for studying the inflammatory changes of smooth muscle fibres, especially in the broad ligament, or in tubal peritonitis; in the latter, we almost invariably find the muscle wall of the tube more or less invaded.

The first change in muscle fibres is that the nucleus becomes granular, divides, and there is an increase of living matter. Next, the whole muscle fibre is enlarged and reduced to protoplasm, the coarse granules arranging themselves in rows, representing inflammatory corpuscles; the small amount of cement substance between the muscle fibres dissolves, and gradually the boundary lines between the spindles disappear and the whole bundle is reduced to inflammatory corpuscles.

It is interesting to watch these progressive changes, the clearly lined muscle fibres, filled with protoplasmic masses, and ready to split into lumps. Sometimes the whole inflammatory process or destruction of muscles is clearly seen or illustrated in acute oöphoritis. In this

we frequently find extended fields of intense acute inflammation, the muscle fibres disappearing or breaking up into granular matter or inflammatory corpuscles, some fibres filled with coarse granules or melting into or partly lost in large lumps of living matter; while the remains of muscle fibres scattered here and there remind one of broken pieces of plank on a wide expanse of waters.

Amid the sea of inflammatory corpuscles may occasionally be seen a bright, shining hæmatoblast—a mass of living matter, taking up the hæmoglobin from the blood, on its way to forming red blood-corpuscles. In such intense inflammation a few newly formed red blood-corpuscles are always found, and near by are frequently seen the solid or vacuoled tracts of living matter, marking out the already forming blood-vessels; while in other fields may be seen new blood-vessels winding along gracefully, showing that even in intense inflammation nature is making plans for repair. These may be the future blood-vessels of the new tissue that will grow out of the protoplasmic mass, if the connections of living matter are not destroyed. Muscle fibres when once destroyed can no more be renewed or formed again. They are lost forever. Fibrous connective tissue, being always the tissue of repair, takes its place, thus causing more or less cirrhosis. Frequently, in case of chronic or sub-acute oöphoritis, large portions of the ovary are replaced by a growth of fibrous connective tissue. An ovary so changed can no longer perform its normal functions, and must to some extent be a source of distress. In like manner the muscle fibres of the Fallopian tubes may be replaced by fibrous connective tissue, the mucosa altered, the epithelia gone, and the once highly organized structure loses its fine organization, and is no longer capable of performing its full physiological functions.

Duranté and others studied the muscle changes in endarteritis, and it was shown that muscle spindles break up into rows of inflammatory corpuscles, which afterward change to fibrous connective tissue.

In inflammation the smooth muscle fibres are transformed into highly glistening lumps of living matter, appearing either homogeneous or with a varying number of vacuoles. Many of these muscle spindles coalesce into large masses of living matter after liquefaction of the intervening basis substance. In transverse sections of these muscles we meet with multinuclear protoplasmic bodies which have originated from fused muscle spindles. (See Fig. 3.) These multinuclear heaps break into inflammatory corpuscles, which closely resemble those arisen from previous connective tissue.

In milder forms of myositis the inflammatory changes are confined to, or principally take place in, the delicate fibrous connective tissue, between the groups of smooth muscle fibres. A limited number of muscle fibres may be broken up into inflammatory corpuscles, and these, with the corpuscles sprung from the connective tissue, may remain interconnected, and as in plastic peritonitis, will lead to the augmentation of the interstitial connective tissue. This process also is seen in certain forms of oöphoritis, when the cortex of the ovary will show a considerable increase of fibrous connective tissue, and a diminution of the muscle fibres.

In interstitial salpingitis we often find the most intense form of myositis. Almost all of the muscle fibres may become inflamed, and be transformed into embryonal and medullary matter, sometimes not a vestige of the original muscle fibres can be found.

Plastic Peritonitis.—Inflammatory corpuscles sprung from previous fibrous connective tissue, endothelia, or epithelia, or smooth muscle fibres, remain, as a rule, interconnected by means of delicate offshoots traversing the light interspaces between the inflammatory corpuscles. While this intercommunication remains undisturbed, the inflammatory process is termed "formative" or "plastic." After the subsidence of the inflammation the newly formed inflammatory corpuscles proceed to the formation of new tissues. The inflammatory corpuscles be-

come first elongated, afterward spindle-shaped, and by a reformation of the basis substance develop new connec-

FIG. 5.—Peritoneal Pseudo-membranes. (× 50.) *P*, cord-like pseudo-membrane, with arteries, veins, and capillaries; *O*, dense fibrous connective tissue on the surface of the ovary, the result of plastic oöphoritis.

tive tissues or pseudo-membranes. In patients who have suffered from tubal disease, and have had in consequence repeated attacks of peritonitis, frequently the uterus or uterine appendages, or both, are not only firmly bound down but almost buried in a mass of strong, well-organized pseudo-membranes. Such conditions necessarily produce much suffering, and must shorten life.

Pseudo-membranes, when examined under high powers of the microscope, are of exceeding interest (Fig. 5).

I have chosen for illustration pseudo-membranes originated from the surface of an ovary in a mild process of acute peritonitis. The ovary in this case was transformed into a blood cyst, and buried in all directions in dense adhesions, which obviously had resulted from repeated attacks of peritonitis. The septic matter from the tubes had probably caused not only the peritonitis but also the disease of the ovaries. Nearly all of the normal structure of the ovary was destroyed by the inflammatory process. The outcome of peritonitis, combined with superficial oöphoritis, was in this case a transformation of the outermost layer of the cortex into dense fibrous connective tissue abundantly supplied with blood-vessels, and but scanty vestiges of previous muscle fibres could be seen.

The covering epithelium of the ovary, as stated above, behaves in inflammation exactly like the endothelia of the peritoneum. The surface of the ovary is frequently found with papillary projections, giving to the naked eye an opaque velvety appearance. These projections are the outcome of mild attacks of peritonitis, superficial portions of the ovarian structure having changed to fibrous connective tissue.

The pseudo-membranes are made up of coarse bundles of fibrous connective tissue slightly interlacing. (See Fig. 6.) In any specimen we will see longitudinal as well as transverse and oblique sections of bundles. Between them we will find a varying amount of protoplasmic bodies, the more numerous when the pseudo-membranes

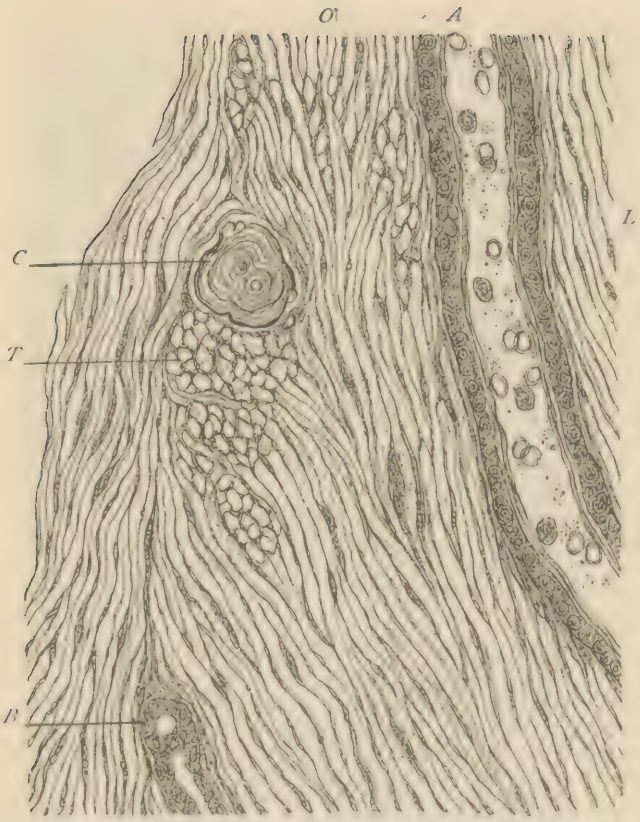

FIG. 6.—Peritoneal Pseudo-membrane. (× 500.) *L*, longitudinal bundles of fibrous connective tissue; *T*, transverse bundles of fibrous connective tissue; *O*, oblique bundles of fibrous connective tissue; *C*, amyloid corpuscle; *A*, artery; *B*, capillary blood-vessel.

are of recent date. Embedded in this tissue are a varying number of blood-vessels, arteries, arterioles, veins, and capillaries. Most of the blood-vessels are newly formed.

The arterioles are characterized by the presence of a single layer of smooth muscle fibres in the middle coat. Their endothelial layer is, as a rule, pronounced and somewhat irregular. The capillaries, when newly formed, have large, irregular endothelia. In old pseudo-membranes the newly formed blood-vessels have assumed all of the features characteristic of normal ones. Often in pseudo-membranes are found amyloid or colloid corpuscles of a peculiar high power of refraction and a pronounced concentric stratification.

The newly organized connective tissues, or pseudo-membranes, were called by the humeral pathologists "exudates," and according to their views the exudates arose from the dilated blood-vessels of so-called serous membranes, which became organized and produced pseudo-membranes or adhesions, hence the term of plastic exudates is still in use by pathologists and clinicians. This view was successfully contradicted some forty years ago by Virchow.[1] We have seen clearly that the pseudo-membranes are formed by changes of living matter. They are not exudates from dilated blood-vessels, but are firmly and densely organized fibrous connective tissue, and which can hardly be absorbed by blisters on the external skin.

Frequently we find in the pseudo-membranes fields of acute inflammation. A large extent of surface may be thus invaded, or even the whole mass filled with inflammatory corpuscles. Such inflammatory attacks cause the increase of old pseudo-membranes and increased hyperplasia of the peritoneum.

In the pseudo-membranes are also frequently found foci of suppuration or miliary abscesses, as seen in Fig. 7.

Do not these suggest the possible danger of leaving

[1] A late text-book says: "The pathology of pelvic peritonitis is, first, sub-serous congestion, followed by an exudation of plastic material or the higher organized constituents of the blood. Small blisters applied to the iliac region, and repeated, favor the absorption of the inflammatory products" (Skene).

pseudo-membranes, either after removing organs or when simply separating the adhesions and allowing the

FIG. 7.—Miliary Abscess in the Peritoneal Pseudo-membrane. (× 100.) *F*, dense fibrous connective tissue; *N*, bundles of non-medullated nerve-fibres; *A*, artery; *V*, vein.

organs to remain *in situ?* These foci of suppuration necessarily become sources of great peril. Besides, if organs are sufficiently diseased to produce such dense

adhesions, in most instances there is an utter destruction of their physiological functions, and, if the organs are allowed to remain, will produce new or similar adhesions.

Purulent Peritonitis.—When the original inflammatory corpuscles break asunder, we have a formation of pus corpuscles or purulent peritonitis. Under the most favorable circumstances the pus may become encysted in dense, fibrous connective tissue—harmless temporarily, but constantly a threatened danger and a continued source of irritation—and will give more and more trouble until the pus finds some outlet.

Localized superficial suppuration of the peritoneum, on account of its frequent occurrence and serious import, becomes a matter of exceeding interest. The conditions which exist in such localized ulcerations of the peritoneum are illustrated in Fig. 8.

We see the endothelia in the shape of large, multinuclear bodies, derived from the effusion of several endothelia. These multinuclear bodies first become separated into inflammatory corpuscles, then become isolated and changed to pus corpuscles. The pus corpuscles are embedded in an albuminous exudate, and in specimens are visible, as a rule, only in small numbers along suppurating surfaces, since they have become detached and are no longer objects of cutting by the section cutter.

The suppuration may extend deep into the peritoneum and even into the adjacent organs. In one instance, which I reported in the Medical Record, August, 1886, almost the entire ovary was destroyed by the suppuration or by an abscess. The patient, when brought to the hospital, was in an almost dying condition from purulent salpingitis. The uterine appendages were removed, and thereby the patient's life was saved, and she is, to this day, in excellent health.

Etiology of Peritonitis.—Traumatism is uniformly placed as one of the causes of peritonitis, but by far the most frequent cause is sepsis or infection. A cut or tear in the peritoneum, when there is no sepsis, heals rapidly;

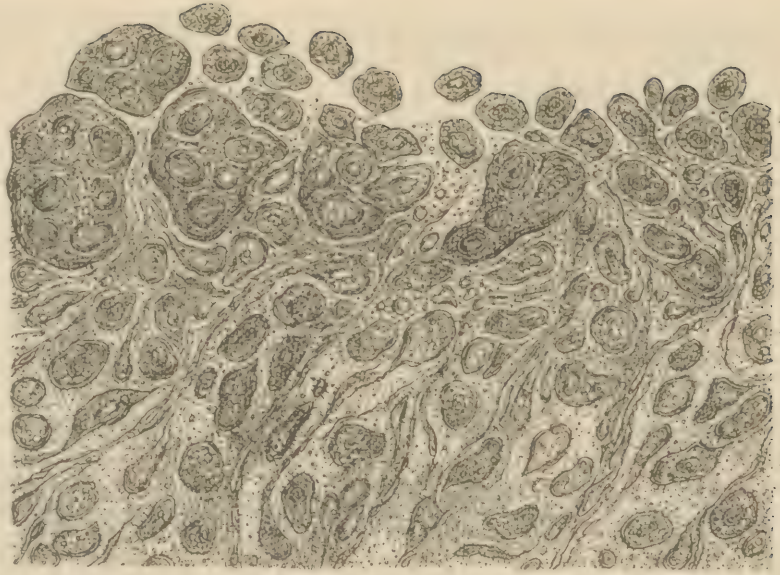

FIG. 8.—Suppurative Peritonitis of the Tube. (× 1,000.) *M*, multinuclear protoplasmic bodies, arising from endothelia; *P*, pus corpuscles, isolated protoplasmic bodies; *I I*, inflammatory corpuscles; *B*, basis substance reduced to protoplasm.

the surgeon can scarcely keep the cut surfaces apart. Extensive wounds or the severest trauma may be inflicted on the peritoneum and no peritonitis result, as tearing away adherent uterine appendages, removing large ovarian cysts, or the operation of supra-pubic or kolpo-hysterectomy. In February, 1888, I removed a uterus, enlarged by a myoma, weighing thirteen and a half pounds; then through the vagina, the stump, with the attached fibroids, thus severely wounding the peritoneum, yet the patient had no peritonitis, nor any special rise of temperature. She was up on the fifteenth day, and since has been in good health. In the same year a patient consulted me who had retroflexed and retroverted uterus, and it, with the uterine appendages, was bound in with extensive and well-organized membranes to the pelvic floor, forming on the right a large abscess which extended into the adjoining aponeurosis, the wall of which abscess was partly formed by the remains of the right ovary and tube. Only a trace of the calibre of the tube could be seen. The whole diseased mass, with the adherent uterine appendages, was removed, destroying considerable portions of the peritoneum, yet, with all this traumatism, the patient made a rapid recovery, and had no peritonitis. Adherent to the specimen were some fibres of the psoas and iliac muscles.

In another case there was a fluctuating tumor bound to the floor of the pelvis. This tumor was removed, thus tearing away a large portion of the peritoneum, besides which, the bleeding was so severe at the ragged and adherent edges of the wound that it was necessary to clamp with forceps; yet there was no peritonitis. The peritoneal cavity, as in the former case, was flushed out, and still further cleansed by being packed with gauze made aseptic by heat.

It was afterward ascertained by microscopical investigation that the growth removed in the last-named patient was a cancer of the most malignant type, and the cancerous material had infiltrated the adjoining tissues; yet

with all these unfavorable circumstances cleansing the peritoneum prevented peritonitis, and the patient made a good recovery.[1]

Pathologists generally agree that the presence of some micro-organisms is necessary for the production of peritonitis. We will find that it is the pyogenic bacteria, as staphylococci and streptococci, that caused the inflammatory abscesses and death. If kept chemically pure, no tissue heals more rapidly or can safely stand more injury than the peritoneum. We see how it can endure, with apparent impunity, for years, the continued irritation of a uterus enlarged by intramural fibromata or sub-peritoneal growths.

In severe trauma, when perceptibly there is no source of infection, the rupture of the veins, arteries, and capillaries may set free pathogenic germs, and cause sepsis and peritonitis. It is a well understood fact that the blood contains various micro-organisms, as staphylococcus pyogenes aureus (Rosenbach) or streptococci citreus, albus, and flavius, discovered and described by Passet. Ogston says: "Staphylococci cause diffuse suppuration, while streptococci are found in circumscribed abscesses."

According to the researches of Senn and others these microbes, while kept in circulation, do no harm, and may be destroyed in the system or eliminated by the excretory organs. Even pure cultures may be injected directly into the circulation, or into the peritoneal cavity, and no peritonitis ensue; but if there is a wound in the peritoneum, or any part of the body, and thereby the circulation is stopped, the microbes in the blood become localized and a soil is thus afforded for the development of the germs. A person may have tubercle bacilli in the blood, and for years be in apparent good health, yet an injury to the bone may cause a localization of the bacilli, and osteomyelitis or tuberculous infection of the joints may de-

[1] Subsequently the cancer returned, but the operation very much prolonged the patient's life.

velop. So syphilitic germs may be in the blood, ready to develop some adverse condition whenever there is a soil or localization. Whatever may be the germ of syphilis, nothing is more deadly or more apt to produce fatal results in case of an operation.

Another cause of peritonitis, or source of infection, may be an ulcerating surface in a distant part of the body, from whence the pus microbe may be conveyed.

Again, various micro-organisms are often introduced by unclean fingers, instruments, etc., especially after confinement, when the abrasions and tears in the cervix and the uterine mucosa are as yet destitute of the protecting epithelium at the previous site of the placenta. This is especially the source of much trouble, and has no doubt destroyed the health and lives of an untold number of women, as so many ignorant and uncultivated midwives assume the duties of the office without previous preparation, many of whom have not the least idea of antisepsis or surgical cleanliness. Every one, man or woman, who does the work of an obstetrician should understand the principles of asepsis and antisepsis. The records of well-conducted maternity hospitals show the very great saving of life from carefulness in these respects. We could gather no such lessons from the millions of isolated cases unrecorded. The large mortality of women at the child-bearing period tells the story.

So far as is at present known, the most frequent pyogenic germs of peritonitis are gonococci. Noeggerath observed clinically the continued contagiousness of gonorrhœa, even after an apparent cure of months' and years' standing, and he suggested the plausible theory of "latent gonorrhœa," which, from a clinical stand-point, was the best presentation. Scientific investigation has shown the foundation for this theory, and proved that the gonococci do remain in the system for years, and may travel to remote parts or organs. Dr. Ely [1] mentions the record of nine cases of malignant endocarditis, in which

[1] Proceedings of the New York Pathological Society, 1888, p. 160.

the urethra appears to have been the point of entrance of the micro-organisms. Bergman found gonococci in pus removed from the knee-joint three weeks after the beginning of arthritis. The continued and irrepressible presence of these gonococci explains how the disease may be communicated years after an apparent cure.

Soon after infection the gonococci are found in the mucosa of the uterus, and may develop gonorrhœal endometritis in the healthiest woman. Then follows gonorrhœal or purulent salpingitis; the mucosa of the tubes becomes intensely inflamed; the delicate folds increase in size, and are filled with inflammatory corpuscles; they commence to suppurate; the suppurating process may extend into and destroy the muscular walls of the Fallopian tubes; and pus may find its way into the peritoneal cavity, producing repeated attacks of suppurative peritonitis. I believe gonorrhœal infection is one of the most frequent causes of disease of the uterine appendages, and of inflammations and suppurations of the peritoneum. Nature, for temporary protection, may seal up the fimbriated extremity, but there is constant danger, and the "pus tubes" may at any time produce a rapidly fatal peritonitis.

The fearful consequences of these diseases, whether produced by gonorrhœal infection or from the introduction in various ways of staphylococci or streptococci, should urge us to consider some mode of protection. No disease is more serious in its possible results. No greater calamity can happen to a woman. It blights forever her dearest hopes, cruelly darkens many of her brightest visions, and destroys forever the organs that make her a woman, and by which she may become a mother. The removal of the diseased uterine appendages by surgery is only to save her from more seroius possibilities. With or without an operation she is sterile.